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REPORT DOCUMENTATION PAGE

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1. REPORT NUMBER	2. GOVT ACCESSION NO. / 3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle): Dental Alloys: Biological Considerations,	5. TYPE OF REPORT & PERIOD COVERED: Manuscript for publication
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): E. Huget (10) Eugene F. Huget	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS: U.S. Army Institute of Dental Research, Walter Reed Army Medical Center, Washington, D.C. 20012	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: Review Manuscript
11. CONTROLLING OFFICE NAME AND ADDRESS: US Army Medical Research and Development Command (11), ATTN: (SGRD-RP, WASH., DC 20314	12. REPORT DATE: 11 Jan 1977 13. NUMBER OF PAGES: 36
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office): (12) 37 p.	15. SECURITY CLASS. (of this report): UNCLASSIFIED 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

DISTRIBUTION STATEMENT A
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Distribution Unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

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18. SUPPLEMENTARY NOTES

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19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Dental Alloys, nickel, chromium, beryllium, cytotoxicity, neoplasia, contact dermatitis.

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The usefulness of nickel-chromium based alloys in military dental practice stems mainly from their demonstrated physical and mechanical properties. However, sufficient clinical experience for elucidation of any risks attendant to the intraoral use of these materials is lacking.

The amount of avialable literature which attests to the abilities of nickel and chromium to produce acute and chronic states of disease in man is awesome. To date, however, substantial data and clinical experience which would unequivocally contraindicate the use of castable dental alloys formulated from

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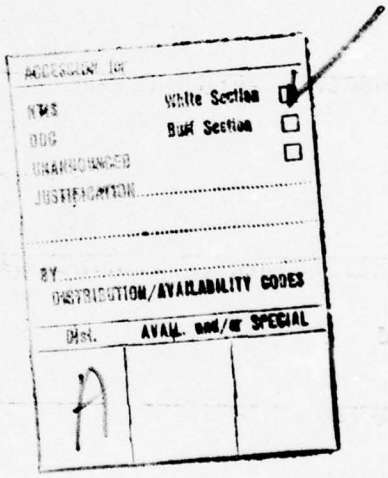
these elements are nonexistent. Conversely, the present lack of sufficient, appropriate and reliable long term biological data has precluded definitive demonstration of the safety of the base metal alloys.

Competent scientists must assess, as objectively as possible, the risks incurred under the actual conditions of intraoral use of base metal alloys. On the other hand, others must estimate the benefits that could possibly arise through the use of these materials. It is likely that research relevent to these matters will increase in the near future. Hopefully, the high standard of integrity that has characterized research activity in dental materials will be maintained.



DENTAL ALLOYS: BIOLOGICAL CONSIDERATIONS

Eugene F. Huget

Division of Dental Materials

U.S. Army Institute of Dental Research

Washington, D.C. 20012

Commercial materials and equipment are identified in this report to specify the experimental procedure. Such identification does not imply official recommendation or endorsement or that the equipment and materials are necessarily the best available for the purpose.

In recent years, costs related to the laboratory fabrication of cast fixed dental restorations have increased markedly. Increased production costs have been, for the most part, manifestations of current gold prices. Searches for less expensive materials from which inlays, crowns and bridges might be cast have led to interest in the use of materials alloyed from base-metals rather than from precious constituents. Today, more than 20 base-metal alloys are available for clinical use.

The clinical usefulness of base-metal crown-and-bridge-alloys stems mainly from their demonstrated physical and mechanical properties. However, sufficient clinical experience for elucidation of any risks attendant to the intraoral use of these materials is lacking. It would appear, from the paucity of biological data, that neither the efficacy nor the safety of the base metal alloys can be ascertained adequately.

It is generally accepted that risks are facts of life. Risks related to the health and welfare of man are endless: The risk of contracting carcinoma of the lung through inhalation of the combustion products of tobacco; the risk of accidental injury through participation in a contact sport; the risk of not remembering the date of a memorable occasion. Some risks are easily perceived, some are nebulous and some we conceal from either ourselves or others. Ultimately, however, certain risks must be accepted whether willingly or unwillingly, whether knowingly or not.

Clinicians and dental scientists alike have little or no concept of the absolute level of risk associated with the use of many common restorative materials. It is unlikely that such insight would be useful, except perhaps in choosing between equally convenient alternatives of widely different risk.

Metals and Neoplasia

During the past decade, the concern of environmentalists and scientists to identify cancer-producing substances has become emense. It would appear, from television, newspaper and magazine reports, that exposure to cancer producing agents is unavoidable. Although the so-called cancer experts do not agree completely as to which substances are hazardous, more than 1,400 carcinogens have been identified on the basis of animal tests. However, fewer than 25 of these substances have been shown to be carcinogenic to man.

Compositional features of base-metal restorative alloys point to the need for careful investigation of their potential for the production of untoward adverse tissue reactions. Compositions of the "economy" alloys are, in essence, departures from the compositions of surgical implant and partial denture casting alloys. Nickel (60% to 80%) and chromium (12% to 20%) are the major constituents of most available products. Unfortunately, nickel[1-10] and chromium [11-12] are known to have a limited but definite carcinogenic potential in man. Nickel, at the present time, is listed among the ten most common carcinogens.

Malignant neoplastic disease of the lung and nasal sinuses is found with alarming frequency among workers engaged in the production

of nickel-containing alloys.[13-16] Documented case histories of patients afflicted with nickel-induced malignancies include chronic, long term industrial exposure to the etiologic agent as a causal factor. In addition to the carcinogenic hazards of nickel inhalation for industrial workers, nickel carcinogenesis may constitute a risk for the general population. Nickel has been detected in the gaseous phase of cigarette smoke, suggesting that nickel may be one of the carcinogenic constituents of tobacco.[17-18] Certain nickel compounds have been shown to be carcinogenic to rodents when administered by inhalation or by parenteral routes. Mitchel[19] implanted pellets of a nickel-gallium restorative material subdermally in Wistar rats and found that sarcoma developed at one or more implantation sites in 9 of 10 animals. Five of 10 rats which received implants of pure nickel also developed sarcoma. However, no malignant lesions developed in any of 10 other experimental groups of 10 rats each, which received implants of a diverse selection of other dental materials.

The presence of nickel in implanted prosthetic devices may also present a possible carcinogenic hazard for man. McDougall[20] has described the clinical course of a sarcoma that developed in the soft tissue of the arm 30 years after implantation of a steel plate. Also, Dube and Fisher[21] have reported the development of a hemangioendothelioma in the tibia and soft tissues after implantation of a steel prostheses. In both patients, the implanted devices were fabricated from an alloy which differed from that of the anchoring screws. Implantation of metals that are of dissimilar composition

usually results in unnecessary electrolysis and corrosion. Dube and Fisher have speculated that metallic corrosion products, including nickel and chromium, were responsible for the induction of the hemangioendothelioma in their patient.

Modern living is accompanied by seemingly endless opportunities to encounter nickel. As one nickel manufacturer has claimed, "nickel is with you from the time you get up in the morning until you go to sleep at night."[22] Although certain malignancies have been attributed to chronic exposure to nickel, the routine daily use, handling and wear of nickel-containing items have never been implicated in the production of neoplastic disease.

Beryllium Disease

Beryllium,[23-27] a metal which is toxic in the free state, as well as when combined with phosphorous, oxygen, fluorine or sulfur, is also a constituent of a few available alloys. The toxicity of beryllium and its compounds was recognized in Germany as early as 1933.[28] Acute toxicity is characterized by irritation of mucous membranes and occasionally by acute pneumonitis with either a fatal outcome or complete remission.[29-30] Chronic beryllium disease affects the respiratory system, with varying histologic patterns of interstitial granulomas and cellular infiltration, restriction of lung volume, hypoxemia, and radiographic findings consistent with the pathologic features. Latency in the onset of symptoms from time of first exposure may range from 5 to 24 years.[27]

Response of a beryllium sensitive individual to beryllium compounds can be progressive and severely destructive. In small

doses beryllium is excreted unchanged in the urine, but in slightly larger doses it reacts with tissue proteins. In highly sensitive individuals, a progressive granulomatous response may be aroused by the presence of amounts causing only some fibrosis in the majority. The reaction, which can progress without further exposure, is significantly different from those exhibited by people sensitized to other metals and their compounds.[26]

The diagnosis of beryllium disease is based on two sets of criteria, one epidemiologic, the other clinical. The epidemiologic criterion is a significant beryllium exposure, namely, an exposure to beryllium or its toxic compounds, and one which has produced similar disease in others. Clinical criteria include (1) diffuse densities on roentogenograms; (2) patterns of respiratory insufficiency; (3) interstitial granulomatous pneumonitis; (4) systemic toxicity demonstrated by functional or pathologic abnormalities in tissues other than lungs; and (5) beryllium in tissues. A diagnosis of beryllium disease requires significant exposure and the presence of at least the first two clinical criteria.

Hazards from exposure to beryllium arise mainly from melting, grinding and finishing procedures. Inhalation of beryllium-containing fumes and dusts is the major route of exposure. Standards for the safe management of beryllium-containing alloys have been promulgated by both the National Institute of Occupational Safety and Health (NIOSH) and the American Dental Association.[31] These standards emphasize the importance of adequate general and local exhaust ventillation for all laboratory procedures in which beryllium-containing materials are employed. The maintenance of rigorous regimens of personal and laboratory hygiene is also stressed.

In view of the fact that dusts and fumes which contain nickel are carcinogenic to man, standards applicable to the handling of beryllium-containing materials should probably be enforced in production dental laboratories where any nickel-based alloy is used.

Metals and Hypersensitive Reactions

Disease other than neoplasia may result from contact-exposure to metals.[32-38] Relatively noble and precious metals as well as base-metals have been linked to the production of allergic contact dermatitis. This disease-entity represents a delayed hypersensitive reaction to a contact allergen that has provoked an immune response. If any area of the skin becomes sensitized, no matter how small, the entire body surface is sensitized.

Sensitization to a contact allergen is predisposed by many factors. Burned, infected or eczematous skin destroys the natural epidermal barrier and permits an increased incidence of contact dermatitis. Previous sensitization to one allergen may predispose to sensitization to another allergen or allergens if the allergens are immunochemically related substances.

Capacity to react to an allergen varies from person to person. Some individuals are more susceptible at certain times than at others. Usually, the very young and very old are affected less than others. This finding may reflect either a lower incidence of exposure or a diminished ability to react.

The clinical picture of allergic contact dermatitis is characterized by the appearance of erythema, papules and edema. Small vessicles may also develop. Severe reactions may exhibit large weeping blisters.

Gold is generally regarded as a relatively inert and safe material. However, the belief that gold is nonsensitizing is not substantiated by literature reports. Allergic dermatitis caused by the wearing of a gold ring has been reported by Comaish.[39] Stomatitis from a gold dental restoration with concomitant dermatitis at sites contacted by gold jewelry has been described by Elgart and Higdon.[40] Sensitivity of a patient to a 14-karat gold orbital prosthesis has been reported by Forster and Dickey.[41] Considering the number of individuals who are exposed to gold jewelry and gold dental restorations, the number of confirmed cases of gold-sensitivity is extremely low.

Documented cases of platinum-dermatitis and palladium-dermatitis are even more rare. Platinosis is caused not by metallic platinum, but by contact with complex platinum salts.[42] Mainly, the disease affects platinum refiners. Cutaneous manifestations of platinosis which may include pruritus, erythema, eczema and urticaria are usually limited to exposed parts of the body. One case of palladium sensitivity in a laboratory worker engaged in research on precious metals has been reported by Munro-Ashman, et al.[43]

Chromium compounds are recognized for their ability to induce contact dermatitis as well as for their potential to cause severe corrosive irritation of skin.[44] Both effects are common in industrial exposure. In the general population, however, the allergic type of dermatitis is found almost exclusively.[45] Exposure to chromates[46-52] may occur through the handling or use of detergents, bleaches, shaving creams, lotions, chrome-tanned leathers, matches,

yellow and orange paints, hide glues and chromated catgut. However, metallic chromium, chromium-containing alloys and chromium-plated objects do not produce allergic contact dermatitis in chromium-sensitive individuals. Dermatitis that results from contact with a chromium-type alloy is usually due to the presence of metals other than chromium.

In the United States, as well as abroad, nickel ranks third among the 5 most common causes of allergic contact dermatitis.[53] In the general population, nickel dermatitis is found more frequently among women than among men. Nickel dermatitis of the earlobe is common in nickel-sensitive women. Signs and symptoms of sensitivity may become manifest even upon contact with nickel-containing gold jewelry. With changes in modern eye wear fashions and the concurrent increased popularity of metal-framed spectacles, another means by which prolonged contact with nickel can be made has become available.

Nickel containing metallic implants have been linked to the production of sensitization dermatitis. Also, the surgical removal of implanted nickel-containing orthopedic devices and missile fragments has resulted in the clearing of dermatitis in nickel-sensitive patients.

The ability of an alloy to induce a dermatitis appears to be related to its pattern and mode of corrosion.[54-56] All base metal implants corrode in the presence of tissue fluids.[57] Corrosion of nickel and chromium containing implants, with the exception of those fabricated from stainless steel, facilitate the migration of nickel and chromium to the surrounding tissues. Nickel, however, does not appear to be selectively leached from surgical-grade stainless steel.

Nickel dermatitis may spread, in a symmetrical fashion, to secondary sites.[58] Occasionally, associated areas of dermatitis appear to bear no relationship to the primary site of eruption. Secondary sites may include the arm, eyelids and sides of the neck and face.

Mechanisms involved in the spread of nickel dermatitis to distant areas are not understood. However, such spreads may be more apparent than real. It is probable that so-called secondary sites are contaminated by prespiring fingers during the initial eruptive stage.

A simple, inexpensive and reliable test developed by Fleigl[59] can be used to detect the ability of metal objects to produce a contact dermatitis in nickel-sensitive individuals. The placement of 2 to 3 drops of a 1 percent alcoholic solution of dimethylglyoxime and a few drops of ammonia water on a metallic object, on skin or in a solution will produce a strawberry-red insoluble salt in the presence of available nickel. Most nickel-containing alloys yield a positive test. Stainless steel, however, is a noted exception.

Unpolished as-received ingots of nickel-based dental alloys as well as unpolished castings fabricated from these materials yield a positive test for available nickel.[60] A positive test is not obtained, however, with highly polished ingots and castings. This would suggest that available nickel is striped from the test-surfaces or that impervious protective films of debris are deposited on the metallic objects during the polishing procedure.

In vitro investigations have revealed that some nickel-based alloys do not passivate.[61-62] When compared to dental golds, these materials exhibit relatively high rates of corrosion. It is conceivable that products resulting from the corrosion of base metal dental restorations could trigger a soft-tissue inflammatory response and thereby provide opportunity for the initiation of an irritancy or sensitization dermatitis. Also, the placement of a corrodable cast restoration in an area in which gingival tissues have been damaged iatrogenically or improperly managed may prompt sensitization of a susceptible patient.

Regardless of alleged corrosion resistance, nickel-containing alloys do not appear to be the materials of choice for use in the mouths of patients who present histories of nickel-sensitivity.

Dental Alloys: Toxicological Studies

In the past, toxicological studies on dental restorative materials have been concerned mainly with acute effects. In principle, acute toxicity can be ascertained readily by appropriate animal-laboratory tests or by other techniques. Results of a 3-month biocompatibility study conducted by Moffa,[63] revealed that subcutaneous implants of nickel-chromium alloys in rabbits were as well tolerated as a gold alloy control. On the other hand, Sandrick, Kaminski and Greener[64] found that nickel-based implants were encapsulated by fatty degenerative tissue, whereas controls of 316L stainless steel showed only mild tissue reaction and the usual fibrous connective tissue capsule.

A recent animal-implant study conducted at the United States Army Institute of Dental Research compared tissue responses elicited by two nickel-chromium based crown-and-bridge alloys (Ticon* and Gemini II+) with those produced by two surgical-grade base-metal casting alloys (Vitallium# and Ticonium§).[65]

Constituents of the as-received alloys, with the exception of carbon, were determined quantitatively by atomic absorption spectrophotometry. Carbon contents were determined by combustion gravimetric techniques. Significant variations among the alloys with respect to nickel and chromium content were found (Table 1). Neither Surgical Vitallium nor Surgical Ticonium contained beryllium. Beryllium content of Gemini II was greater than that of Ticon by a factor of approximately 4.

Implant specimens were 3 mm by 0.5 mm-discs cast by conventional dental laboratory procedures. The castings were honed to remove investment and oxide coatings. Then the discs were polished manually on 240 to 600 grit abrasive papers, cleaned ultrasonically in water and autoclaved.

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* CMP Industries, Albany, N.Y.

\+ Kerr Mfg. Co., Romulus, MI.

\# Howmedica Inc., Chicago, ILL.

§ CMP Industries, Albany, N.Y.

A disc of each alloy was implanted subcutaneously in the abdomens of 24 male albino rats. Four animals were sacrificed at each of the following post implant periods: 3 days; 1 week; 2 weeks; 4 weeks; 12 weeks and 1 year. At each sacrifice, the discs and surrounding soft tissues were harvested and placed in 10 percent buffered formalin. After a minimum fixation period of 1 week, the tissues were cut from the metal discs. Paraffin sections were prepared, stained with hematoxylin and eosin and examined microscopically.

At 3 days, tissues affiliated with the four alloys showed a moderate inflammatory infiltrate characterized by polymorphonuclear leukocytes in a fibrin mesh. Signs of acute inflammation were less apparent at 1 week. However, all one-week specimens contained a well-vascularized granulation tissue. Orderly deposition and alignment of collagen fibers were salient features of all tissues harvested at post-implant periods of 2 weeks and 4 weeks. The connective tissue walls of 12-week tissue preparations were well defined. Nuclei of the fibrocytes diminished with respect to both size and number. At 1 year, the walls of all samples were significantly less cellular. The remaining nuclei appeared to be distributed throughout the collagenous material. Inflammation was not a feature of one-year tissue specimens.

From histologic findings, differences between reactions of the subcutaneous tissues of the rat to the four casting alloys could not be discerned. The generic nature of the responses suggests that encapsulation may be either a manifestation of rejection or merely a reparative response to injury. These findings appear to

be in agreement with those of other investigators whose work has indicated that the subcutaneous biocompatability of some nickel-containing alloys is not significantly different from that of a dental gold.[63]

The results of such studies, with regard to the relative safety of nickel-based dental alloys, tend to be encouraging. Today, however, there is increased concern about the biological responses that may not become apparent until several years after infiltration and absorption of a potentially toxic substance.

Animal experiments, for a number of reasons, can not be tailored to yield critical data required for the fail-safe determination of long-term human experience. First of all, species differences may be very marked and conclusions based on a comparison of one animal species with another may be misleading. Secondly, the life-spans of different species of animals are very different. Lastly, long-term animal experiments are costly and difficult to perform.

Feasibility of the use of tissue culture techniques for determination of the cytotoxic potentials of base metal casting alloys has also been investigated at the United States Army Institute of Dental Research. A recent study assessed cellular response to a nickel-based partial denture alloy (Ticonium-100*), to two nickel-chromium crown-and-bridge materials (Gemini II+ and Victory#) and to an iron-chromium based alloy (Dentillium-CB§).[64]

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* CMP Industries, Albany, N.Y.

\+ Kerr Mfg. Co., Romulus, MI.

\# Unitek Corp.,Monrovia, CA.

§ Codesco, Inc., Philadelphia, PA.

Two types of patterns were used to produce castings of the test alloys (Fig 1). All patterns were invested in a phosphate-bonded refractory material.** The molds were placed in a cold gas oven, heated slowly to 1,300F and held at temperature for 45 minutes. The alloys were cast into heat-soaked molds with the use of an automatic induction casting machine.++ Casting temperatures for Ticonium 100, Gemini II, Victory and Dentillium-CB were 2,580, 2,700, 2,600 and 2,800F, respectively. After breakout, the castings were liquid-honed to remove investment and oxide-coatings.

One series of patterns yielded discs with approximate dimensions of 1 mm X 6 mm. The discs were cut from their sprues and polished manually on 240-600 grit abrasive papers.

Castings produced from other patterns were sectioned to obtain 8-mm diameter spheres and 35-mm X 3-mm rods. The cast pieces were cleaned ultrasonically. Sixty spheres and a like number of rods of each alloy were churned in a 5-liter capacity ceramic vessel that contained 500 ml water. Rotational speed of the vessel was 60 rpm. Churning-time for spheres and rods of each alloy was seven days.

Upon completion of the wet-milling of each alloy, particulate matter and water were decanted from the ceramic vessel into evaporating dishes. After removal of water by evaporation, residual materials were brushed from the dishes, collected on watch glasses and dried for 14 days at 230F.

** Ceramigold Investment, Whip-Mix Corp., Louisville, KY.

++ Electromatic casting machine, Howmet Corp., New York, N.Y.

Powder-products produced by wet-milling were pressed into 1-mm X 6-mm discs by the following procedure. Approximately 50 mg powder and a steel plunger were placed in a 6-mm X 6-mm steel mold. The assembly was compressed on a constant strain rate testing machine.## Crosshead speed of the machine was 0.02 in per minute. Packing pressure required to produce discs of the desired size was 85,000 psi.

Positive controls for evaluation of cytotoxicity were made from reagent grade Cu_2O.§§ Approximately 150 mg powder were used to form each of 20 compacted discs (~1 mm X 6 mm). Negative controls were pressed discs (~1 mm X 6 mm) of gold foil¶ and discs (~1 mm X 6 mm) of polyethylene.Π

All discs were sterilized by exposure to radiation (100,000 R) from a Co_{60} source.

The surface components of cast and pressed discs of each alloy were determined by X-ray dispersive analysis.

Instron Universal Testing Machine, Instron Corp., Canton, MA.

§§ Allied Chemical Corp., Morristown, N.J.

¶ Pure Gold Cylinders, Williams Gold Refining Co., Inc., Buffalo, N.Y.

Π Vacu-Press Discs, Dentsply International, York, PA.

ΠΠ Nikon Inverted Phase Microscope, Nippon Logaky, Tokyo, Japan.

Monolayer cultures of L-929 mouse fibroblasts and HeLa cells were established in 60-mm diameter plastic petri dishes following the techniques of Guess[67] and Rosenbluth.[68] Each dish contained approximately 1.4×10^6 cells. Triplicate control and pressed powder specimens of each alloy were planted by direct application on cultures of L-929 mouse fibroblasts. All discs applied directly to the cells were stabilized by 6-mm diameter glass rods (Fig 2). An additional series of specimens consisting of controls and powders obtained from Ticonium 100, Gemini II and Dentillium-CB were placed on agar-overlaid cultures of L-929 cells.

The cultures were observed macroscopically and microscopially[Ω] at 24 and 48 hours for assessment of cell alteration and for measurement of zones of lysis.

X-ray dispersive spectrums of cast and pressed specimens are shown in Figures 3 and 4, respectively. Major components of cast-specimens of Ticonium 100, Gemini II and Victory were nickel and chromium. Major components of Dentillium-CB were iron and chromium. Minor constituents of the cast discs included molybdenum, manganese, aluminum, cobalt and silicon. Compositions of the pressed discs were remarkably different than those of the alloys from which the powders were derived. Powders obtained by wet-milling exhibited large amounts of silicon. Other compositional differences between the two types of specimens were reflected by increased amounts of aluminum and by the presence of minor amounts of phosphorus, sodium, chlorine and calcium in the pressed discs.

Ω Energy Despersive X-Ray Analyzer, model 707A with EDAX EDIT II 7 EP Program, EDAX International, Prairie View, IL.

Responses of the cell cultures to the controls and test materials are summarized in Table 2. Cuprous oxide elicited severe cellular changes. At 24 hours, cultures of fibroblasts as well as cultures of HeLa cells exhibited relatively large (18-20 mm) zones of lysis. Cells outside the major zones of lysis showed morphological changes (rounding). Lysis of all altered cells was complete at 48 hours. Gold foil and polyethylene caused neither alteration nor lysis of cells.

Powders obtained from the nickel-chromium based alloys induced cytological changes. Responses to Ticonium 100 tended to be mild. Direct application of powder-discs to HeLa cells and to mouse fibroblasts yielded relatively small (~4 mm) zones of total lysis. Larger (~4 mm at 24 hours and 8 mm at 48 hours) primary zones of lysis were produced by placement of pressed Ticonium 100 specimens on agar-overlaid cultures of L-929 cells. Responses to Gemini II were moderate. HeLa cells appeared to be more sensitive to pulverized constituents of the alloy than were mouse fibroblasts. Responses to Victory were severe. HeLa cells and mouse fibroblasts were affected similarly. At 24 hours, zones of total lysis (18-20 mm) were significantly larger than those produced by the other nickel-chromium based materials. Approximately 30 percent of all cells situated between the primary rings of cell destruction and the peripheries of the plates were lysed. All remaining cells exhibited rounding and loss of stain. Lysis of all cells was complete at 48 hours.

Powders obtained from Dentillium-CB as well as cast discs of all four test materials caused neither alteration nor lysis of the cultured cells.

It is likely that wet-milling produced materials somewhat akin to both the wear and corrosion products of the four casting alloys. Contamination of the milled products by silicon, aluminum, phosphorus, sodium, chlorine and calcium is attributed to inclusion of finely ground particles of the ceramic milling-vessel. However, these contaminants did not appear to be involved in the production of adverse cellular changes.

Tissue reaction to a metallic material is believed to be related to the number and species of ions released into the tissues. The failure of all cast discs to evoke an unfavorable tissue response suggests that potentially toxic ionizable components are not available at the specimen surfaces. The finding would further indicate that the cast metals are passive. However, in time, and while in contact with tissue, ionization of the components of any alloy can occur. Severity of the ensuing tissue reaction will depend upon the cytotoxic potentials of ions made available through dissolution of products produced by wear and corrosion of the alloy's surface.

In contrast to the behavior of the iron-chromium powder derived from Dentillium-CB, it would appear that powders of Ticonium 100, Gemini II and Victory readily release toxic ions. Beryllium, a potentially toxic element, is known to be a constituent of Ticonium 100 and of Gemini II. On the other hand, beryllium is not a constituent of Victory. Beryllium and other elements with atomic

numbers lower than that of sodium (atomic number 11) can not be detected by the analytical method employed in this study. Other compositional features of the test materials implicate nickel in the production of adverse cellular reactions.

Numerous uncertainties cloud the clinical significance of data based on the response of cultured cells to the milled products of nickel-containing casting alloys. Factors which include the rate of release of ions from the in-service base metal dental restoration as well as the number and species of ions absorbed by the target tissues of the human host remain to be determined.

Summary

The amount of available literature which attests to the abilities of nickel, chromium and beryllium to produce acute and chronic states of disease in man is awesome. To date, however, substantial data and clinical experience which would unequivocally contraindicate the use of castable dental alloys formulated from these elements are nonexistent. Conversely, the present lack of sufficient, appropriate and reliable long term biological data has precluded definitive demonstration of the safety of the base metal crown-and-bridge alloys.

Clinical application of the new base-metal restorative materials would appear to require acceptance of risk by both the dentist and the patient. The quantitative level of risk for a single patient may be of less importance than the attitudes of the dentist and of the patient -- their anxiety or nonchalance -- with regard to risks in general. To an individual, a new risk may make only a meager addition to the other risks encountered in every day living. In considering the wide use of nickel-based alloys in the general

population, however, risk evaluation should have a rather different and much greater significance. While the risk to an individual may be trivial, when affecting a large segment of the population it may imply a considerable increase in morbidity.

Competent scientists must assess, as objectively as possible, the risks incurred under the actual conditions of intraoral use of the base metal crown-and-bridge alloys. On the other hand, others must estimate the benefits that could possibly arise through the use of these materials. It is likely that research relevant to these matters will increase in the near future. Hopefully, the high standard of integrity that has characterized research activity in dental materials will be maintained.

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Legends for Figures

Fig. 1. Patterns for preparation of specimens: (A) Discs (~1 mm X 6 mm); (B) Spheres (~8 mm diameter) and rods (~35 mm X 3 mm).

Fig. 2. Assembly used to stabilize discs applied directly to cultured cells.

Fig. 3. X-ray dispersive spectrums of cast discs: (A) Ticonium 100; (B) Gemini II; (C) Dentillium CB; (D) Victory.

Fig. 4. X-ray dispersive spectrums of pressed powder-discs: (A) Ticonium 100; (B) Gemini II; (C) Dentillium CB; (D) Victory.

Table 1

ALLOY COMPOSITIONS

Element	Surgical Vitallium	Surgical Ticonium	Ticon	Gemini II
	%	%	%	%
Cobalt	61.1	15.4	0.92	0.00
Chromium	31.6	24.6	16.1	12.4
Nickel	0.29	54.3	70.4	80.5
Molybdenum	4.41	4.31	3.96	2.00
Manganese	0.71	0.03	3.77	0.00
Silicon	0.63	0.45	0.42	0.00
Carbon	0.40	0.013	0.033	0.25
Iron	0.58	0.71	0.75	0.13
Aluminum	0.01	0.02	3.96	2.8
Copper	0.01	0.03	0.01	0.00
Beryllium	0.00	0.00	0.48	2.1
Titanium	0.00	0.00	0.01*	0.00
Tungsten	0.00	0.00	0.65*	0.00

* Value includes columbium, tantalum and zirconium if present.

Table 2

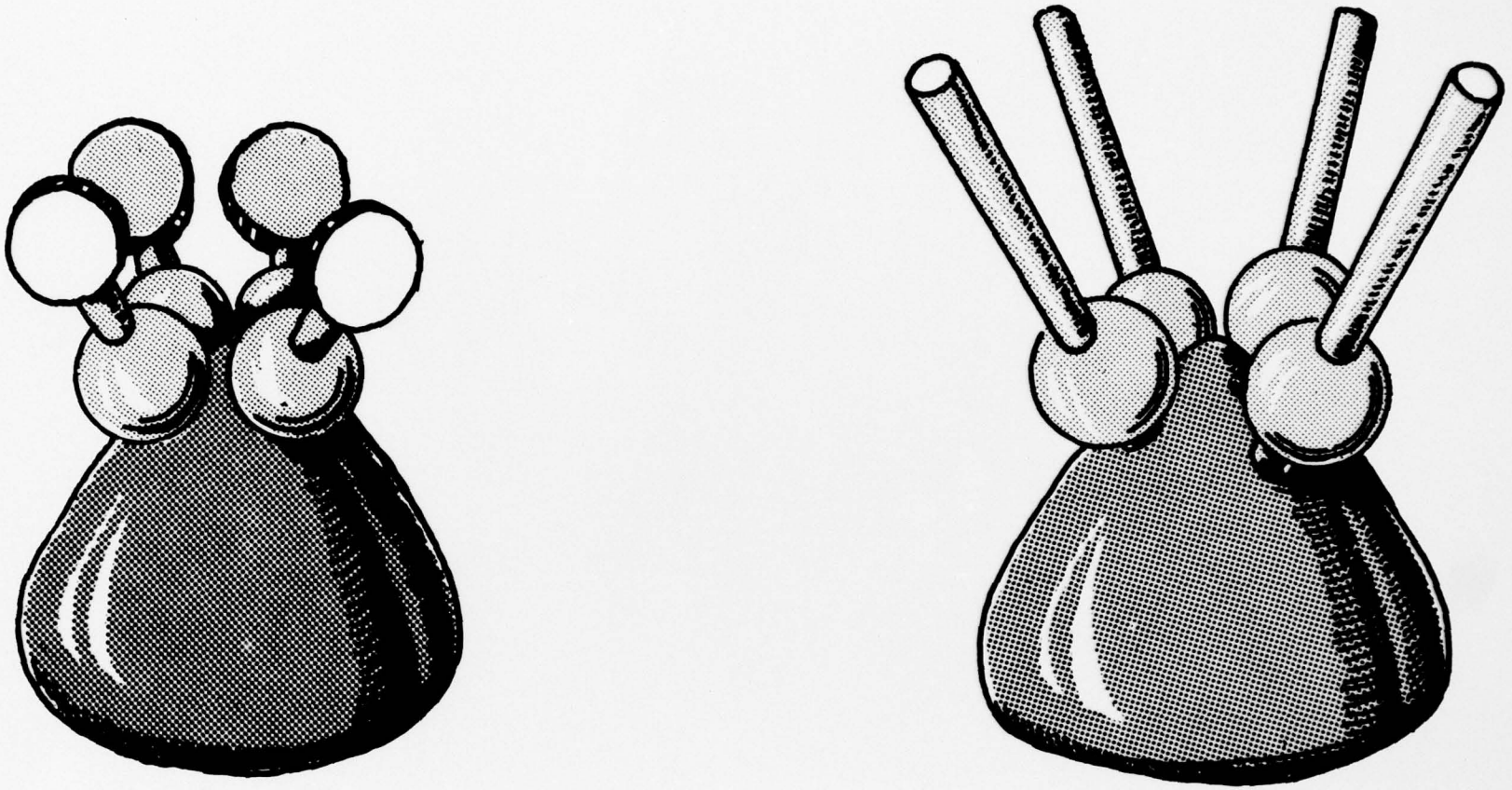
CYTOTOXIC RESPONSES - ZONES OF LYSIS (mm)

Materials	Test Medium: L-929 Fibroblasts 24 hrs	L-929 Fibroblasts 48 hrs	HeLa Cells 24 hrs	HeLa Cells 48 hrs	Agar Overlaid L-929 Fibroblasts 24 hrs	Agar Overlaid L-929 Fibroblasts 48 hrs
Positive Control*	20	∞ ¶	20	∞	18	∞
Negative Control+	nil	nil	nil	nil	nil	nil
Ticonium 100						
As-cast	nil	nil	nil	nil	nil	nil
Powder	1	3	1	2	4	8
Gemini II						
As-cast	nil	nil	nil	nil	nil	nil
Powder	3	12	8	22	5	9
Victory						
As-cast	nil	nil	nil	nil	nil	nil
Powder	20	∞	18	∞	---	---
Dentillium CB						
As-cast	nil	nil	nil	nil	nil	nil
Powder	nil	nil	nil	nil	nil	nil

* Cuprous oxide, pressed discs.

* Polyethylene and gold foil.

¶ Lysis of all cultured cells.



A

B

Fig.1

Fig.2

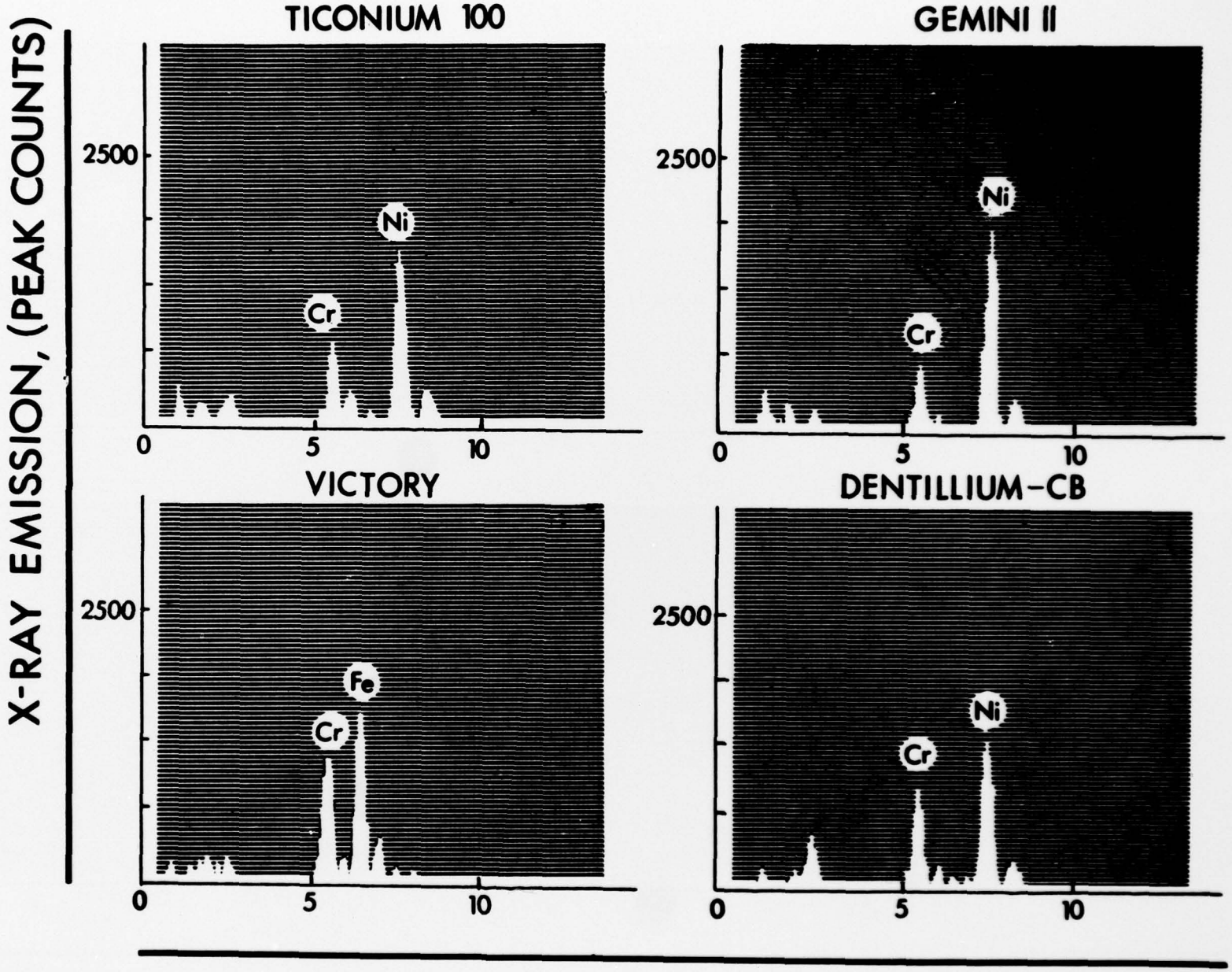

TICONIUM 100
GEMINI II
VICTORY
DENTILLIUM-CB
2500
0
5
10
Ni
Cr
Fe
X-RAY EMISSION, (PEAK COUNTS)
X-RAY ENERGY, keV

Fig.3

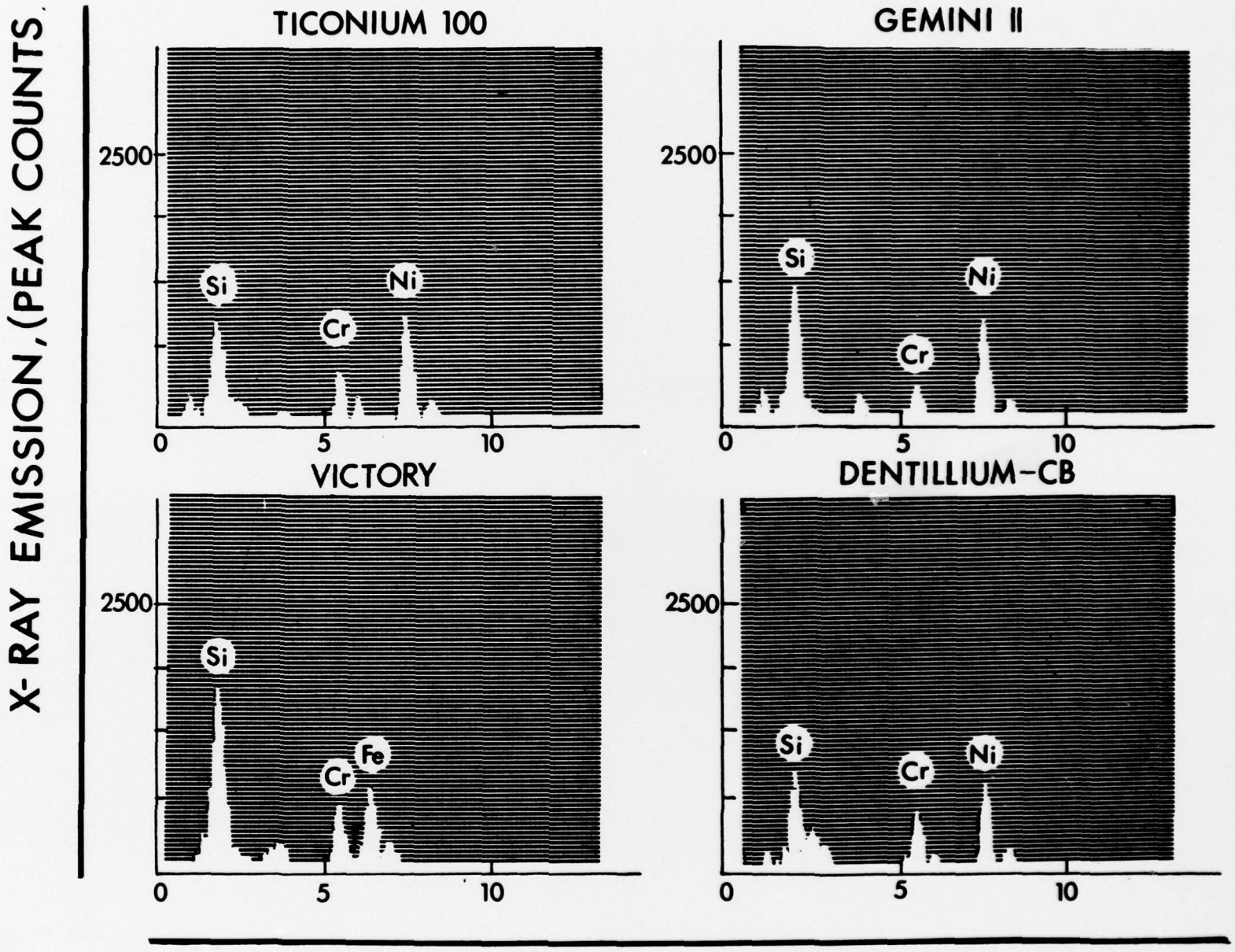

TICONIUM 100
GEMINI II
VICTORY
DENTILLIUM-CB
2500
Si
Ni
Cr
Fe
0
5
10
X-RAY EMISSION, (PEAK COUNTS
X-RAY ENERGY, ke V

Fig.4